

Evaluation of the Role of Oropharyngeal Fatty Infiltration In Obstructive Sleep Apnea Syndrome

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Abstract

Background: Obstructive sleep apnea (OSA) patients owing to oropharyngeal fatty infiltration have a narrower pharyngeal airway than the normal individuals.

Material and Methods: To identify potential fatty infiltration of the pharyngeal tissues that may contribute to OSA syndrome, a histologic study of the distal soft palate was performed on a sum of 100 cases including OSA patients (80 cases) and normal subjects (20 cases) taken as controls. All cases were divided according the body mass index (BMI) into two OSA groups (group 1: normal weight OSA; 25 cases & group 2: overweight OSA; 55 cases) and two control non-OSA groups (group 3: normal weight controls; 10 cases & group 4: overweight controls; 10 cases). All specimens underwent a quantitative analysis of the fatty tissue that showed an excessive fatty infiltration in all oropharyngeal biopsies received from OSA groups compared to a normal infiltration in control biopsies.

Results: No significant correlation was found between the pharyngeal fatty infiltration on one side and BMI or apnea index (AI) on the other.

Absence of an excessive fatty infiltration in the control subjects, irrespective to his or her weight, and its presence in all studied OSA patients concluded that this event may play a role in the upper airway obstruction and can be associated with the development of apnea.

Key words: Oropharynx — Fatty tissue infiltration — Obstructive sleep apnea — Histopathology.

Introduction

Obstructive sleep apnea (OSA), currently recognized as a potentially lethal condition, affects 2–4 percent of adults (Young *et al.*, 1993; Olshansky *et al.*, 2005). It is defined as repeated complete cessation of breathing (apnea) that occurs a minimum of five times per hour of sleep due to an occlusion in the upper airway (Young *et al.*, 2004; den Herder *et al.*, 2004).

Several symptoms are characteristic of OSA syndrome i.e. daytime sleepiness, morning headache, diminished libido or impotence, depression, mood disorders, impaired memory and concentration together with heavy snoring (Erkinjuntti *et al.*, 1990; Ress *et al.*, 2000 & Guilleminault *et al.*, 2003). Airway obstruction may affect

the nasopharynx, oropharynx and hypopharynx, however, the most common site for obstruction is the oropharynx (Victor, 1999; Pennsylvania Patient Safety Authority, 2007).

Certain skeletal structures are characteristic of OSA male patients such as reduced sagittal linear dimensions of the cranial base, maxilla and bony naso- and oropharynx (Ingman *et al.*, 2004; Taylor *et al.*, 2006). Soft tissue factors can also predispose to OSA, for example tonsillar hypertrophy and obesity, which can cause fatty infiltration into the pharyngeal tissues (Strollo and Rogers, 1996; Kollia and Krogstad, 1999 & Shine *et al.*, 2005). In addition to the anatomical factors that result in a narrowed upper airway space,

suppression of the pharyngeal muscle activity in sleep is also critical to OSA by producing a narrower airspace that is more vulnerable to collapse on inspiration. OSA is ultimately caused by the impact of brain sleep mechanisms on the processes that control motor outflow to the pharyngeal muscles, the tone of which is necessary and sufficient to keep the airway space open during wakefulness (Horner, 2001; Donnelly, 2005). In the supine position, during nocturnal sleep, the pharyngeal soft tissues obstruct the pharyngeal airway even more (Ingman et al., 2004).

Cessation of airflow lasting for more than 10 seconds, observed at least 30 times during 6-7 hours of nocturnal sleep (apnea index [AI] of more than 5), or a respiratory disturbance index (RDI) of more than 10 is diagnostic of this syndrome. Decreased arterial blood oxygen saturation, systemic and pulmonary hypertension and severe cardiac arrhythmias during sleep together with coronary artery atherosclerosis as well as an ischemic stroke are cardiovascular changes seen in OSA patients (Daniels, 2001; Sorof et al., 2002; Hanevold et al., 2004; Berenson, 2005; Couch and Daniels, 2005 & Kaw et al., 2006). A decrease in the apnea-or hypopnea-related arterial oxygen saturation of more than 4% is considered pathologic (Zohar et al., 1998; Caprio, 2006 & Choudhary et al., 2007).

Obstructive sleep apnea may be seen in individuals of any age but being more frequent after the age of 40 (Zohar et al., 1998; Young et al., 2002 & Pennsylvania Patient Safety Authority, 2007).

There were agreement among physiologists and physicians, treating OSA patients, that the pharyngeal fatty infiltration in obese ones influences the degree of OSA and has a pathophysiologic role in the initiating and progressing OSA process (Davies and Stradling, 1990; Zerah et al., 1993 & Vgontzas et al., 2005). The degree of overweight is expressed in a term of body mass index (BMI) in kilograms per square meter and the obesity is considered when a BMI is of 25 kg/m^2 or more (Zohar et al., 1998; Hedley et al., 2004; Gould et al., 2006; Suter et al., 2006 & Nader et al., 2006).

The aim of this study is to evaluate the histologic quantitative relationship of the pharyngeal fatty tissue in obese and

normal weight OSA patients compared to that in the normal non-OSA subjects whatever their body weight.

Material and Methods

The material of this study consisted of a sum of one hundred (100) pharyngeal specimens obtained by tonsillectomy and uvulopalato-Pharyngoplasty (UPPP) from Otorhinolaryngology Departments, Al-Azhar University Hospitals during the period from December 2005 to April 2007. The specimens obtained from eighty (80) OSA patients (Table 1); seventy five of whom were males (94.3%) while the remainings (5.7%) were females. The patients' ages ranged from 24 to 63 years old (mean 44.52 ± 2.9). The apnea index (AI) ranged from 12 to 60 and the respiratory disturbance index (RDI) ranged from 20 to 46. The recorded sleep arterial blood oxygen saturation varies from 78% to 84% (mean 79.9%). The average sleep time in OSA groups was 357 minutes, their average sleep time efficiency was 82% and the average sleep time spent in their rapid eye movement was 16 minutes. According to the BMI, two groups of OSA patients were studied; group 1: included twenty five (25/80; 31.2%) normal-weight OSA patients with a BMI ranged from 23.3 upto 24.8 and group 2: involved fifty five (55/80 ; 68.8%) overweight patients with a BMI ranged from 25.2 to 38.2. In addition, twenty (20) specimens were received from normal individuals without OSA or snoring as controls. Their ages ranged from 19 to 40 years (mean 26.31 ± 2.1), fourteen (14/20;70%) of whom were males and the remainings (6/20;30%) were females. The recorded mean sleep arterial oxygen saturation was 96.2%. All control specimens were taken by tonsillectomy in-between the episodes of acute tonsillitis. Regarding the BMI, the control cases were also divided into two groups; group 3: included ten (10/20;50%) normal-weight subjects with a BMI ranged from 22.9 to 24.1 and group 4: involved ten (10/20;50%) overweight subjects with a BMI ranged from 25.1 to 31.9.

Each OSA patient spent the night for an average of 7 hours of testing. The airflow was measured by oral or nasal

thermistors, the arterial oxygen saturation level was recorded by a pulse oximeter and the snoring severity was quantitated in decibels.

The fresh tissue specimens from OSA patients (contained the uvula and anterior or posterior tonsillar pillar) as well as the control specimens (including anterior and posterior tonsillar pillars) were fixed in 10% formalin and subsequently embedded in paraffin. The blocks were sectioned at 5 μ m thickness. The sections were stained with hematoxylin and eosin and examined microscopically. The amount of infiltrated fatty tissue was evaluated by two observers semiquantitatively in 50 consecutive low power fields according to the following grade (Zohar *et al.*, 1998; Table 2).

For more accurate quantitative analysis, the tissue stained sections of all 4 groups were re-examined by the computerized image analysis calculating the mean percentage of the infiltrated fatty tissue surface area in relation to the total examined areas using Image Pro Plus V4.51 (Media Cybernetics Inc. 2002) and the results were evaluated.

The statistical analysis was performed according to the Chi square and student (T) tests to estimate the significance of results using Microsoft Excell XP 2002. The value <0.05 was statistically significant.

Results

Obstructive sleep apnea (OSA) patients (Table 3) displayed mild degree (Fig.1) of excessive fatty infiltration in 32 cases [12 in group 1; 12/25 (48%) and 20 in group 2; 20/55 (36.4%)], moderate degree (Fig.2) in 32 cases [9 in group 1; 9/25 (36%) and 23 in group 2; 23/55 (41.8%)] and severe degree (Fig.3) in 16 cases [4 in group 1; 4/25 (16%) and 12 in group 2;

12/55 (21.8%)]. No significant correlation ($P>0.05$) between the body mass index (BMI) and the degree of fatty infiltration in these groups as the smallest percentage of group 2 patients (21.8%) with the lowest BMI range (25.2-28.4) showed a severe degree of excessive fatty infiltration (Table 3). This result was supported by the presence of insignificant difference ($P=0.28$) between the mean percentage of the infiltrated fatty tissue surface area in both OSA groups (Table 4).

In addition, both control groups (groups 3&4) revealed up to 10% of fatty tissue infiltration (normal degree) in their specimens (Fig.4) irrespective to the BMI values. An insignificant difference ($P=0.11$) was detected between the mean percentages of infiltrated fatty tissue surface area in these groups, however, a significant difference ($P<0.05$) was noticed in these mean percentages of group 1 or group 2 compared to those of group 3 or group 4 (Table 4).

Moreover, we displayed insignificant difference ($P=0.32$) between apnea index (AI) and the degree of excessive fatty infiltration in OSA patients (Table 5). No difference was found between the degree of fatty infiltration of the uvula and that of anterior and posterior tonsillar pillars.

Considering the age, our study detected an insignificant difference ($P=0.16$) between the degree of fatty infiltration and the ages of the control non-OSA individuals. However, OSA patients from 20 to 40 years of age (18/80; 22.5%) showed mild to moderate fatty infiltration degree and the patients more than 40 years of age disclosed all degrees of fatty infiltration & more than 50% of whom (37/62) had a severe degree of infiltration ($P<0.05$; Table 6).

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Table 1: Clinical variables in the studied cases (n=100)

Variables	OSA Patients (n=80)	Control non-OSA (n=20)
* Sex:		
Male	75 (94.3%)	14 (70%)
Female	5 (5.7%)	6 (30%)
* Age (Years):		
Range	24-63	19 - 40
Mean	44.52 \pm 2.9	26.31 \pm 2.1
* Body Mass Index (BMI; Kg/m ²):		
Normal-weight	23.3 - 24.8	22.9 - 24.1
Overweight	25.2 - 38.2	25.1 - 31.9
* Apnea Index (AI)	12 - 60	—
* Respiratory Disturbance Index (RDI)	20 - 46	—
* Mean Sleep arterial O ₂ saturation	79.9	96.2%

Table 2: Semiquantitative evaluation of fatty tissue infiltration (Zohar et al., 1998).

Variables	Degree of Fatty infiltration
Normal:	Up to 10 %
Excessive:	
Mild	10 - 20 %
Moderate	21 - 40 %
Severe	> 40 %

Table 3: Degree of fatty tissue infiltration in OSA patients (n=80):

Degree	Group 1 (Normal-weight OSA) ·			Group 2 (overweight OSA) ·		
	No.	%	BMI (Kg/m ²)*	No.	%	BMI (Kg/m ²)*
Mild	21	48	23.9 - 24.8	20	36.4	29.2 - 38.2
Moderate	9	36	23.8 - 24.4	23	41.8	29.6 - 35.0
Severe	4	16	23.3 - 24.1	12	21.8	25.2 - 28.4
Total	25	100	23.3 - 24.8	55	100	25.2 - 38.2

· OSA = Obstructive sleep apnea;

* BMI = Body mass index

Table 4: The mean percent of the infiltrated fatty tissue surface area in all studied cases (n=100):

	Group 1 (Normal weight OSA)	Group 2 (Over weight OSA)	Group 3 (Normal weight control)	Group 4 (Over weight control)
Average	34.458	35.240	5.899	6.316
Standard deviation	1.27	1.42	0.09	0.13
Group-compared	1 - 2	2 - 3	1 - 3	3 - 4
P value ·	0.28 (NS)*	<0.05 (S) ·	0.11 (NS)*	<0.05 (S) ·

*NS = Non-significant ; · S = Significant

Table 5: Apnea index and degree of fatty infiltration in OSA patients (n=80):

Apnea index (AI)	OSA Patients		Degree of fatty infiltration		
	No.	%	Mild	Moderate	Severe
12 – 20	36	45	12	21	12
21 – 40	28	35	18	12	5
41 – 60	16	20	8	12	0
Total (12 – 60)	80	100	38	45	17

P value = 0.32 (NS= Non Significant)

Table 6: Age-related fatty infiltration in control and OSA groups (n=100):

Groups	Age range (years)	Degree of fatty infiltration				P value
		Normal	Mild	Moderate	severe	
* Control	19 – 40	20(100%)	—	—	—	NS*
* OSA patients:	20 – 40	—	8(10%)	10(12.5%)	—	
	41 – 60	—	10(12.5%)	11(13.8%)	27(33.8%)	
	> 60	—	4(5%)	—	10(12.5%)	S+
	Total	20(100%)	22(27.5%)	21(26.2%)	37(46.3%)	

*NS = Non significant ; S = significant; OSA = Obstructive sleep apnea.

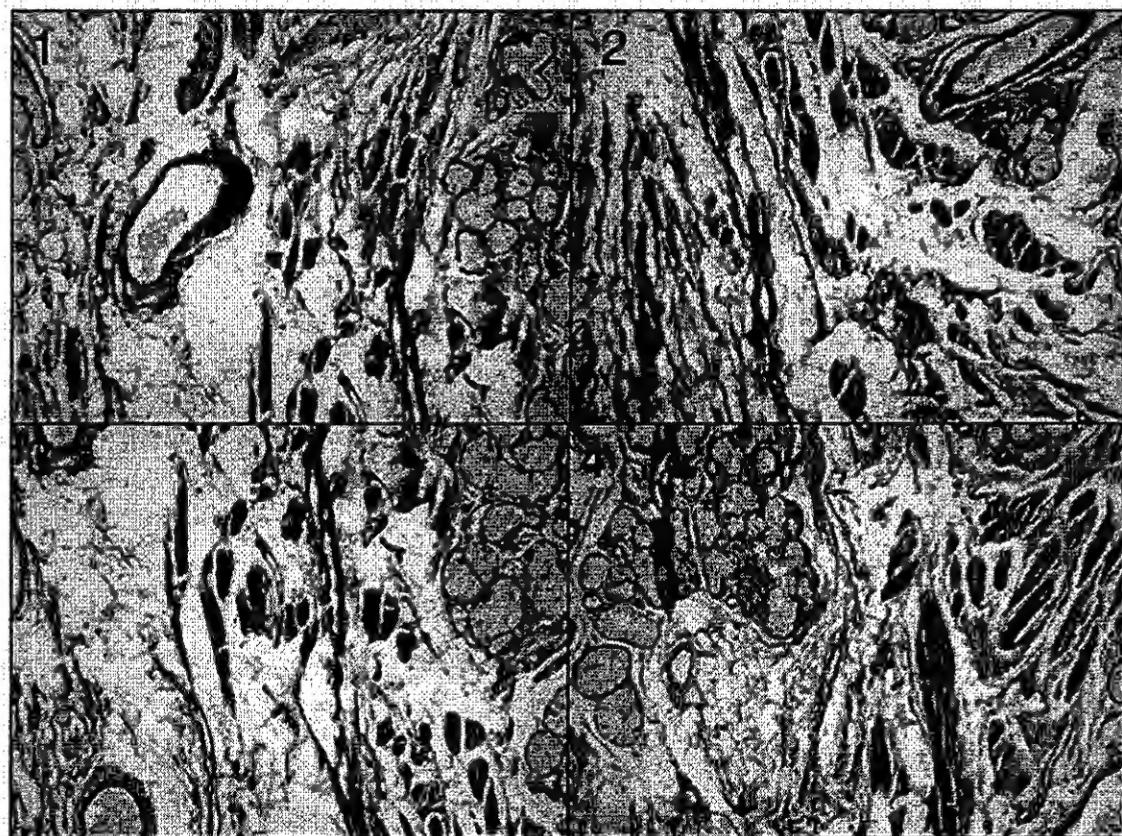


Fig .1 : Oropharyngeal tissue showing a mild degree of fatty tissue infiltration . Fig .2 : Oropharyngeal tissue showing a moderate degree of fatty infiltration . Fig .3 : Oropharyngeal tissue showing a severe degree of fatty infiltration . Fig .4 : Oropharyngeal tissue showing normal degree of infiltrated fat (H x & E x 100)

Discussion

It is uncertainly determined that chronic tonsillitis in the normal non OSA individuals reduces fat in the pharyngeal tissue (Zohar *et al.*, 1998).

We noticed that three in both OSA patients' groups had history of recurrent tonsillitis and their histologic specimens displayed moderate to severe degree of excessive fatty infiltration. This notice agreed with that mentioned by Alvi and Lee, 2005 who found a narrowing in OSA patients upper airway caused by nasal septal deviation, nasal allergies or polyps, enlarged turbinates, an enlarged uvula or low-lying soft palate, retrognathia and micrognathia in addition to the enlarged tonsils.

About 80 to 90% of OSA patients are undiagnosed (Stierer and Punjabi, 2005). The reasons may include inability to recognize sleep-related symptoms as well as lack of time and resources to perform the standard polysomnogram test to diagnose OSA syndrome (Mendez and Olson, 2006). Polysomnography (PSG), being an indispensable evaluative tool for sleep disorders, provides objective data to be integrated into the overall clinical picture, information concerning the type of disorder and its severity as well as the prognostic clues during follow-up period. During PSG, several physiologic variables are recorded while the patient sleeps, including brain electrical activity, eye movements, chin and leg activity, airflow, respiratory effort (i.e. chest and abdominal movement), oxygen saturation and cardiac rhythm (Piccirillo *et al.*, 2000; Olson *et al.*, 2003 & Mendez and Olson, 2006).

The major evaluative criterion in this study was the apnea index (AI) which is the total number of apneic episodes during sleep divided by the number of sleeping hours. An AI greater than 5 is considered abnormal considering with the value detected by (Zohar *et al.*, 1998; Flemons, 2002 & Kaw *et al.*, 2006). Flemons (1999) & Alvi and Lee (2005) mentioned that this abnormal AI value may be associated with excessive daytime sleepiness.

The palatine complex is a muscular structure including the soft palate, uvula as

well as anterior and posterior tonsillar pillars and functioning as a valve to seal the nasopharynx. This flexible structure consists of mucosa, connective tissue, muscle and aponeurosis. The oropharyngeal airway was found to be significantly narrower in the supine position than in the upright position in OSA patients. However, the primary symptom of OSA; apnea, occurs during nocturnal sleep (in the supine position). Thus, the position of pharyngeal tissues in the supine posture is essential in determining the severity of OSA syndrome (Ingman *et al.*, 2004).

We found a significant association between OSA syndrome and obesity as more than two thirds of our OSA patients (68.8%) were overweighed with a BMI exceeding 25 Kg/m^2 . Also, we displayed the prevalence of this syndrome in males representing 94.3%. These findings were similar to those reported Young and Finn, 1998; Zohar *et al.*, 1998 & Finkel *et al.*, 2006; who stated that obesity, independent of the age or hormonal status, is the most common risk factor associated with and may elicit the OSA syndrome causing deposition of fat and increased mass in the uvula and pharyngeal tissues with narrowing of the airways. They also regarded obesity when the person is 20-30% above his or her desirable weight. In addition, Zohar *et al.* (1998); Young and Finn (1998) & Finkel *et al.* (2006) reported not only the prevalence of OSA syndrome in males but also its rare incidence in women.

An insignificant association between the body mass index (BMI) and the degree of fatty tissue infiltration in OSA & control groups was found. Moreover, these data were supported by the detection of insignificant difference in the mean percentages of infiltrated fatty tissue surface area between normal-weight and overweight groups OSA groups and between normal weight and overweight control groups. However, the mean percentages were significantly different in OSA groups compared to the control groups irrespective to the body weight. In addition, we showed insignificant

difference between apnea index (AI) and the degree of excessive fatty infiltration in OSA groups. Furthermore, we noticed no difference in the degree of fatty tissue infiltration detected in the uvula and in either tonsillar pillar. All findings were identical to those observed by *Zohar et al., 1998; Victor, 1999; Finkel et al., 2006 & Pennsylvania Patient Safety Authority, 2007*. Victor (1999) reported that the obese OSA patients have a peripharyngeal fatty infiltration and/or increased soft palate and lingual size. Also, they may have a receding jaw that doesn't allow a sufficient place for the tongue. All these anatomical abnormalities decrease the cross sectional area of the upper airway which together with the diminished airway muscle tone during sleep and the pull of gravity in the supine position impede airflow during respiration.

This study revealed that the degree of fatty infiltration insignificantly changed with the age of the control non-OSA subjects. However, in OSA patients, the degree increased, in severity, with the age being more frequently severe after 40 years old. These data are in concordance with those of *Young et al., 2002 and Pennsylvania Patient Safety Authority, 2007* who stated that OSA prevalence increases with age being higher after the age of 65.

In conclusion, we noticed insignificant correlation between either apnea index (AI) or body mass index (BMI) and the degree of fatty infiltration. Moreover, a value of more than 10% microscopic fatty infiltration in the oropharyngeal tissue may have a potential effect on the pharyngeal compliance and can be associated with the development of apnea.

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تقييم دور التسرب الدهني للبلعوم الفمي في مرض توقف التنفس الإنسيادي النومي

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من أقسام الباثولوجي 1 و الأنف والأذن والحنجرة 2 بطب بنين الأزهر فرعى أسيوط والقاهرة، ومن قسم
الباثولوجي 3 بطب القاهرة

يمتلك مرضى توقف التنفس الإنسيادي النومي مجرى بلعومي أضيق من الأشخاص الأصحاء وذلك بسبب التسرب الدهني للبلعوم الفمي، ولكي نحدد دور هذا التسرب في ذلك المرض فقد أجريت دراسة هستوباثولوجية على مائة فرد منهم ثمانون يعانون من مرض التوقف التنفسى الإنسياد النومي، وعشرون شخص أصحاء أخذوا على سبيل المقارنة، وقد قسمت هذه الحالات حسب وزن الجسم إلى أربع مجموعات: الأولى وتشتمل على 25 حالة من مرض التوقف التنفسى الإنسيادي ذو الوزن الطبيعي، والثانية تشمل على 55 حالة من مرض التوقف التنفسى الإنسيادي يعانون من الوزن الزائد أو السمنة، والثالثة تشمل على 10 حالات أصحاء أوزان أجسامهم طبيعية، والرابعة تشمل على 10 حالات أصحاء أوزانهم زائدة، وقد خضعت الحالات جميعاً لتحليل كمي للنسيج الدهني المتسرّب واتضح أن كميته تزيد في كل مرض التوقف التنفسى عن الأشخاص الأصحاء ولم توجد علاقة ذات أهمية إحصائية بين هذا التسرب الدهني من جانب وبين مؤشر التنفس أو مؤشر كتلة الجسم من جانب آخر، وأظهرت الدراسة غياب هذا التسرب الدهني الزائد في حالات الأشخاص الأصحاء بغض النظر عن أوزانهم مع وجوده في حالات مرضي توقف التنفس الإنسيادي النومي مما يستنتج منه دور هذا التسرب الدهني في انسداد مجرى الهواء العلوي مع إمكانية ارتباطه بحدوث التوقف التنفسى في هؤلاء المرضى.